Correspondence

The Editorial Board will be pleased to receive and consider for publication correspondence containing information of interest to physicians or commenting on issues of the day. Letters ordinarily should not exceed 600 words, and must be typewritten, double-spaced and submitted in duplicate (the original typescript and one copy). Authors will be given an opportunity to review any substantial editing or abridgement before publication.

Ischemic Bowel Infarction and Phenylpropanolamine Use

TO THE EDITOR: Phenylpropanolamine (PPA), a sympathomimetic structural analogue of amphetamine, has been the subject of several recent reports citing amphetamine-like adverse side effects. Temporal association of ischemic effects involving the coronary,[1] renal[2] and cerebral[3] vasculature has been previously reported. To our knowledge, compromise of the visceral vascular bed has not been described.

Report of a Case

A 45-year-old woman presented to our emergency room with a 24-hour history of progressive crampy abdominal pain and bloody diarrhea. The pain had initially been ill-defined and periumbilical but had more recently become well localized to the right lower quadrant and had become increasingly severe. The patient had been in excellent health before the onset of her pain, and her medical history was remarkable only for an appendectomy as a child. Specifically, there was no history of cardiac, rheumatologic or atherosclerotic vascular disease. She was not taking oral contraceptives but had been regularly using a proprietary diet aid, Dexatrim (65 mg of phenylpropanolamine hydrochloride per capsule), for three years. Her initial dosage was low, one to two capsules a day, but she had increased the dosage to three to four capsules per day over the two months before admission in an effort to increase weight loss for her upcoming wedding. At this time, her weight was 60 kg and her height 157 cm.

On presentation, the patient appeared acutely ill. She was febrile and dehydrated. On physical examination, hypoactive bowel sounds and moderate abdominal tenderness that was maximal in the right lower quadrant were noted. There was grossly bloody stool in her rectum; proctoscopic examination showed normal-appearing rectal mucosa with blood from above 25 cm.

Findings on cardiovascular and dermatologic examinations were normal. Abdominal x-ray studies showed an ileus pattern with air-fluid in the left upper quadrant. An x-ray study of the chest and an electrocardiogram showed no abnormalities. Findings on a complete blood count were notable for a leukocyte count of 18,000 per μl with a left shift. Results of SMA-6 and urinalysis were within normal limits.

Over the 12 hours following admission and initial resuscitation, the patient became hemodynamically unstable and peritonitis developed. She then underwent emergent exploratory laparotomy. Operative findings included dark, foul-smelling peritoneal fluid and an obviously ischemic proximal colon with necrosis of a segment of midtransverse colon. There was no gross evidence of either arterial or venous obstruction. An extended right colectomy was carried out with an ileostomy and transverse colon mucous fistula. Gross examination of the resected specimen showed mucosal necrosis. Histopathology was consistent with ischemic colitis, and there was no histopathological evidence for a vasculitic or atherosclerotic etiology for the ischemia.

The patient was advised to refrain from further use of diet pills. One year later she underwent ileocolonic reanastomosis without complication. She has continued to do well without recurrent abdominal complaints.

The cause of the colonic ischemia in our patient is uncertain. There is no evidence of embolic or thrombotic disease, and other reported causes (oral contraceptives, vasculitis, amyloidosis) were not present. As a structural analogue of amphetamine, PPA may have had a role in the ischemic bowel infarction in our patient. Methamphetamine abuse has been shown experimentally to lead to fibrinoid necrosis in intracerebral vessels with the formation of microaneurysms. The mechanism for these vascular changes is thought to be a necrotizing angiitis that is indistinguishable from periarteritis nodosa.[4] This may also involve renal as well as other visceral arteries. Radiologic evidence of cerebral vascular spasm has been reported in one animal study where amphetamines were injected through carotid vascular catheters.[5] Although we cannot document a definite causal relationship between PPA and the colitis seen in our patient, the temporal relationship is suggestive.

Phenylpropanolamine products are widely used in decongestants, anorexiants and "legal stimulants." With the increasing use of this drug, the list of adverse reactions appears to be correspondingly lengthening. The knowledge that a patient has used this drug may be of historical importance in evaluating otherwise unexplained ischemic visceral events.*

LCDR DAVID A. JOHNSON, MC, USNR
Division of Gastroenterology
Naval Hospital
Bethesda, MD 20814-5011
LCDR PERRY W. STAFFORD, MC, USN
Department of Surgery
CAPT ROCCO J. VOLPE, MC, USN
Department of Gastroenterology
Naval Hospital
Portsmouth, VA 23708

*The opinions and assertions contained in this paper are the personal views of the authors and are not to be construed as official policy of the US Department of the Navy or the US Department of Defense.

REFERENCES

1. Pentel PR, Mikell FL, Zavoral JH: Myocardial injury after phenylpropanolamine ingestion. Br Heart J 1982 Jan; 47:51-54

2. Duffy WB, Senekjian HO, Knight TF, et al: Acute renal failure due to phenylpropanolamine. South Med J 1981 Dec; 74:1548-1549

3. Johnson DA, Etter HS, Reeves DM: Stroke and phenylpropanolamine use (Letter). Lancet 1983 Oct 22; 2:970

4. Citron BP, Halpern M, McCarron M, et al: Necrotizing angiitis associated with drug abuse. N Engl J Med 1970 Nov 5; 283:1003-1011

5. Rumbaugh CL, Bergeron RT, Scanlan RL, et al: Cerebral vascular changes secondary to amphetamine abuse in the experimental animal. Radiology 1971 Nov; 101:345-351

Munchausen Alert

TO THE EDITOR: One opportunity to contain rising hospital costs is in early recognition of the "hospital hobo" or Munchausen patient. To remind and alert physicians, we describe a case admitted to Pacific Presbyterian Medical Center, San Francisco, on October 15, 1984.

A 29-year-old, obese white woman walked into the emergency room with the chief complaint of her jaw "going out to the right" while drinking a carbonated beverage. Her jaw had been fractured, she reported, one year before in a fight with a girlfriend. She stated that on her way to our hospital she suffered a seizure, witnessed by friends, and was afraid that her jaw was fractured again. In addition, her surgeons last year (she could not say who or where) elected to stabilize the fragments with rubber bands instead of conventional wires, so she expressed concern that the jaw had not healed properly. She told the emergency physician that her seizures began at age 15 after a head trauma from a fall in gym class. Her current medications were carbamazepine (Tegretol), 200 mg three times a day, and valproic acid (Depakene), 250 mg three times a day.

While having mandible x-ray films made, the patient had another seizure, witnessed by a technician, who considered it to be grand mal epilepsy. She was then sent to the intensive care unit. There she told physicians that she last saw her local neurologist a month ago; she was in San Francisco for a vacation. Past complications included eight respiratory arrests during seizures and two cardiac arrests when phenytoin (Dilantin) was given by rapid intravenous push. Allergies included penicillin, aspirin, codeine, diazepam (Valium) and tetanus toxoid. Her father had seizures, also due to a head trauma, and her daughter died at five months of age due to respiratory arrest from febrile seizures. Her occupation was that of a truck loader. Finally, she had also had two exploratory laparotomies after motor vehicle accidents (the last in May 1984), several venous cutdowns, a cesarean section, a ganglion cyst removal and mild asthma.

Physical examination on admission revealed anisocoria (the right pupil 1 mm greater than the left), a small parietal hematoma and tenderness of the left temporomandibular joint. Neurological examination was significant only for weakness in the right hand and forearm, allegedly due to "nerve damage" from the cutdowns. Her left ankle was in a cast, reportedly placed after a fracture. Her general hygiene was poor, especially dental hygiene, and many teeth were missing.

The patient continued to have seizures throughout the night. Her nurse recorded seizures after a monitor alarm went off, after having foot pain and after being touched on the hand. There was also a seizure after the bedside light was turned on, at which time she stated that her seizures were easily triggered by bright lights and loud noises. Between such episodes, she smoked cigarettes and drank liquids through a convenient aperture in the teeth on the right side of her mouth.

We first saw her about 12 hours after admission. Knowing the valproate level was only 8.4 μg per ml (the therapeutic range 50 to 100), we asked if she had been taking her medication. Through clenched teeth and a jaw jutted to the right, she spoke with little difficulty, telling us that the drugs were taken faithfully. Her neurologists wanted to keep her levels subtherapeutic because she became ill very easily. Use of several other medications in the past had not been successful, including phenobarbital, she said. Yet, before her second motor vehicle accident (May 1984) she only had seizures every two to three months. After the accident they increased to every other day. In addition, she was unconscious for two days after the accident and later, because of severe abdominal pain, underwent an exploratory laparotomy which revealed "three ruptured ovarian cysts." A computed tomographic scan of her head was normal. She then remembered having had two additional seizures while in the ambulance coming to the hospital, but it was noted that she had actually walked in alone.

On our physical examination, palpation of the temporomandibular joints caused no pain when she was distracted. Among her abdominal surgical scars, there was one with a fresh excoriation due to a "stitch abscess." While checking her pupillary response to light, however, she started to blink and stare off to the right. This was followed by tonic-clonic movements of the arms and head. After the seizure, she was unarousable, but returned to her previous state within four minutes. An electroencephalogram was obtained an hour later. It showed no seizure activity, nor any sign of postictal depression, although some mild abnormalities were noted.

That night, the patient signed out against medical advice. The intensive care unit staff had tried to verify, but could not, the existence of her neurologist in central California. When requestioned about this person, she had become furious. It had also been determined by the surgical consultation and x-ray studies that the jaw was not dislocated. We felt that she had been deliberately deviating the mandible. In addition, phenobarbital, which she had denied taking at present, was detected in blood specimens.

With the many inconsistencies in this case, we could only conclude that the patient suffered from Munchausen's syndrome. No doubt other hospital staff will see her in the future. We can supply further details upon request.

TAMMY SMITH
Fourth Year Medical Student
FRED HOM, MD
FORBES H. NORRIS, MD
Department of Neurology
Pacific Presbyterian Medical Center
PO Box 7999
San Francisco, CA 94120

Thrombosis of the Iliac Artery From Seat Belt Injury

TO THE EDITOR: The use of seat belt restraining systems in motor vehicles can protect occupants from major and fatal injuries. Studies have shown a 60% decrease in injuries and a 35% reduction in fatalities by the use of restraining devices.[1,2] An injury caused by a safety belt was first reported by Kulowski and Rost in 1956.[3] This case illustrates a vascular injury incurred from seat belt wear.